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An Aid To Identifying Aspen Diseases Frequently Encountered In The Rocky Mountains

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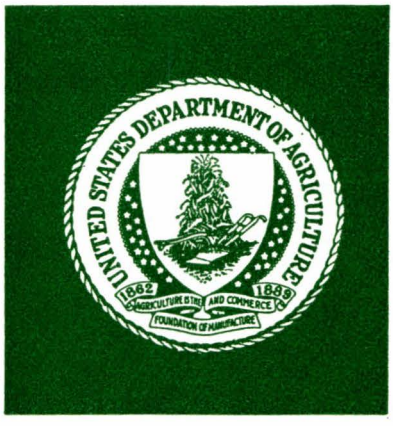

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An Aid To Identifying Aspen Diseases Frequently Encountered In The Rocky Mountains

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Introduction

Quaking aspen, *Populus tremuloides* Michx., is one of the most wide-spread and popular tree species in the Rocky Mountains. Its range extends from British Columbia south through the Western United States into northern Mexico. An aggressive pioneer species, aspen frequently colonizes burns and constitutes the typical fire subclimax of the subalpine forest in the central Rocky Mountains. Aspen frequently reproduces by means of root suckers produced from buds on the lateral roots, usually those within 3 or 4 inches of the soil surface. Over much of its range, aspen is a small to medium-sized, fast-growing, and generally short-lived tree. Under ideal conditions in the West, however, it can attain a height of over 100 feet, a diameter of about 3 feet, and an age of around 200 years.

Long appreciated for its esthetic and shade tree value and its importance as wildlife habitat, aspen is becoming important as a commercial timber species. Although it is capable of good growth and high yields, aspen has one major drawback—its high susceptibility to damage by insects and diseases. The thin, soft, living bark is easily wounded and provides little protection from damage by animals, abiotic factors, and diseases. The most common and damaging types of diseases in aspen are decays and stem cankers; root rots and foliage disease(s) are less common and usually cause only minor damage. The purpose of this guide is to familiarize land managers with some of the most common diseases of aspen in the Rocky Mountains. Estimates of the amount of defect and mortality are included whenever possible. Proper identification of diseases occurring in an aspen stand will help the land manager to write management prescriptions and to better estimate the amount of defect and cull. Because of recent name changes of the various fungi involved, their old, and probably more familiar, names used in other publications are in parenthesis.

A. Aspen trunk rot, caused by the false tinder fungus, *Phellinus tremulae* (Bond.) Bond. et Borris, (*Fomes igniarius* var. *populinus*), is the most common cause of defect and cull in aspen. Fruiting bodies or conks of this fungus are frequently found on stems of living and dead trees and often occur near branch stubs or old wounds. Conks are hoof shaped; the upper surface is gray to black and divided into irregular squares by numerous cracks. The interior of the conk has white flecks and a layered appearance; the color of the lower surface varies from tan to white to dark brown and has many tiny pores. All infected trees do not bear conks, but most trees with advanced decay do have fruiting bodies.

Trees bearing two or three conks (or any conk within 16 feet of the ground) averaged 59 percent cull in a study of aspen in Colorado. Trees with more than three conks above 16 feet were complete losses. This decay is most common in overmature stands; however, it can also infect young trees.

Since infection can occur via wounds, care should be taken to avoid damage to stems during management activities. Shorter rotations and clearcutting will reduce the incidence and impact of this disease.

Figure 1 — *Phellinus tremulae* conks.

B. *Peniophora polygonia* (Pers. ex Fr.) Bourd. et Galz., (*Cryptochaete polygonia*), is probably as common as *Phellinus tremulae* in western aspen; however, this fungus does not form fruiting bodies readily and must be isolated for identification. Since external symptoms are seldom produced by this pathogen, defect or cull it causes cannot be practically evaluated in the field by the land manager. Rot caused by this organism is usually white with a reddish-brown margin. The decayed wood appears more brittle than noninfected wood. Because the incipient stage of decay does not fall out when sawn lengthwise, it is usually considered stained wood and the actual cull attributed to this rot is usually less than that scaled.

A. White mottled rot, caused by *Ganoderma applanatum* (Pers. ex Wallr.) Pat., (*Fomes applanatus*), is quite common in the West and frequently predisposes infected trees to blowdown by rotting the larger roots. Generally the merchantability of infected trees is not seriously affected; fruiting bodies of the fungus, usually found at the base of the tree, indicate extensive butt rot in the lower 4 feet of the trunk. Removal of infected trees is recommended to avoid losses to windthrow and to reduce hazard in recreation sites. Mortality from this disease is minimal in most stands. The white-mottled rot is restricted to the butt and larger roots of the tree. Large, shelflike conks are often formed at the root collar of infected living trees. The conk is smooth, light gray to light brown, and crusty on the upper surface. The lower surface of the conk is white, while the interior is dark reddish brown.

Figure 2 — *Ganoderma applanatum* on a living aspen tree.

Figure 3 — *Ganoderma applanatum* on aspen blowdown.

B. Shoestring root disease, caused by *Armillariella mellea* (Vahl. ex Fr.) Karst., (*Armillaria mellea*), is moderately common in aspen stands. Signs of Armillariella root rot are rhizomorphs, red-brown or black cords of fungus mycelium and white mycelial fans on the roots and root collar. Also, honey-colored mushrooms may appear in late summer or early fall at the base of infected trees. The fungus infects living host tissue, causing decay of the root and butt portion of trees. Mortalilty may occur in patches; however, large numbers of trees are seldom killed.

C. *Flammulina velutipes* (Curt. ex Fr.) Sing., (*Collybia velutipes*), the most frequently encountered butt rot in aspen in Colorado, is responsible for the largest amount of butt cull. The fungus causes a brown mottled white rot which is frequently associated with basal wounds. Decay columns generally average 10 feet in length, except in older trees where they may extend above 16 feet.

Figure 4 – *Armillariella mellea* on aspen.

Figure 5 — *Armillariella mellea* mycelial fans.

Cankers

A. Black canker, caused by *Ceratocystis fimbriata* Ell. & Halst., produces the target-shaped canker frequently seen on aspen trees. The canker is usually associated with wounds on the trunk. Many Ceratocystis cankers are not associated with decay; thus, all trees having a canker should not be considered cull trees. This fungus seldom kills trees, for it spreads only a short distance on the tree each year. In some instances, two or more cankers may eventually kill the tree by coalescing and girdling the stem. Old cankers can weaken the trunk and cause the tree to fail. Prevention of wounds on trees will reduce the incidence of this disease. Severely infected stands should be clearcut and regenerated.

Figure 6 — Target-shaped canker of *Ceratocystis fimbriata;* note annual growth rings of the canker.

B. *Encoelia prulnosa* (Ell. & Everh.) Torkelson and Eckblad, *(Cenangium singulare)* the sooty-bark canker, is aptly named in that a black residue is formed under the infected bark that adheres tightly to the trunk for many years, even after tree death. This canker is more aggressive than Ceratocystis and can cause tree mortality within 4 to 5 years after the initial infection. Spores of the fungus are formed on light-gray, cup-shaped fruiting bodies on the old, dead inner bark. This disease also occurs in association with cambial wounds; the fungus infects trunk wounds and penetrates the inner bark and cambium. The dead outer bark sloughs off 2 or 3 years after infection, exposing the blackened inner bark in an elliptical zonate pattern. Trunk rots are infrequently associated with this canker; thus, many infected trees will yield an acceptable quantity of sound wood. Due to the aggressive nature of this canker-causing organism, felling all infected trees is recommended as a method of preventing rapid stand deterioration.

Figure 7 — *Encoelia pruinosa* canker on aspen.

Figure 8 — Sooty inner-bark of *Encoelia* canker.

C. Hypoxylon canker, caused by *Hypoxylon mammatum* (Wahl.) Miller, is moderately common in aspen stands of the West. At an infection point, the smooth outer bark is sloughed off after several years, revealing a checked cortex. The canker is identified most easily by the laminated, mottled, black and yellowish-white cortex exposed by cutting along the canker margin. Older cankers may be several feet long, and white mycelial fans may be present under the bark. Mortality by girdling can occur on smaller trees within 5 years after the initial infection; however, it may take 20 or more years to girdle larger trees. The merchantability of many infected trees will not be reduced, as decay is not often associated with this canker.

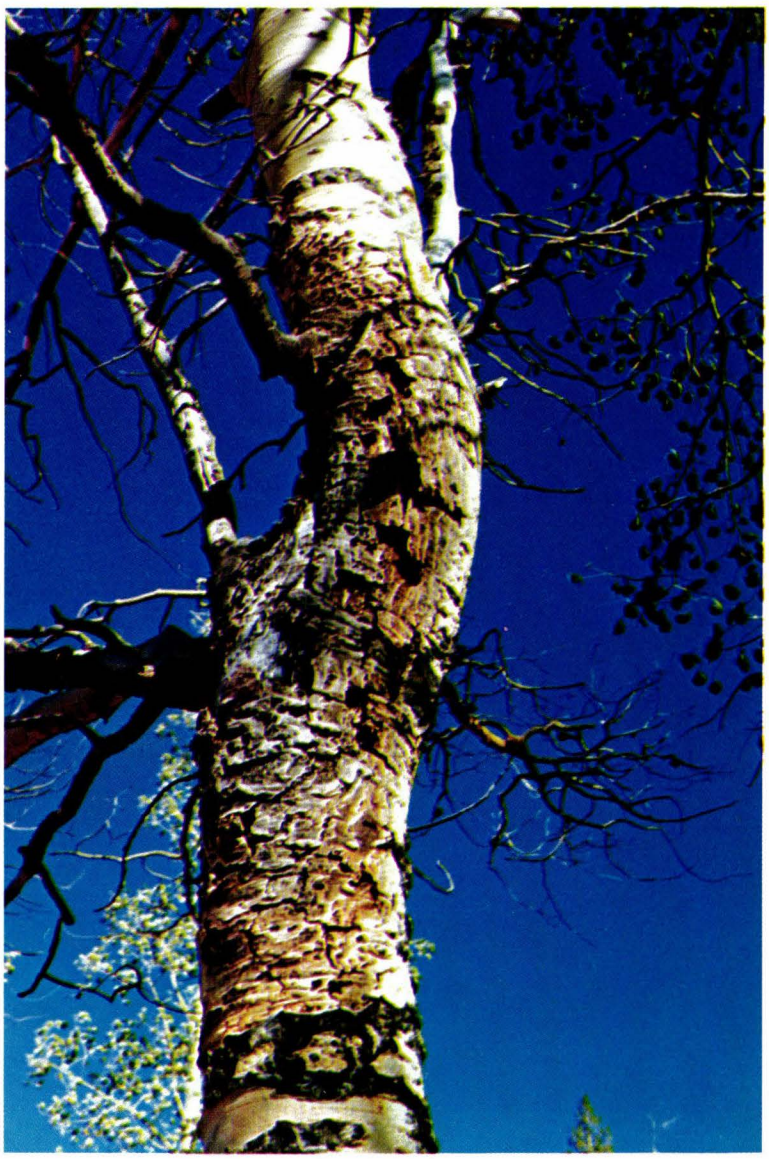

Figure 9 – Relatively young Hypoxylon canker with bark still intact.

Figure 10 — Checked cortex caused by Hypoxylon infection.

D. Cytospora canker, caused by *Cytospora chrysosperma* (Pers.) Fr., is widespread on aspen. Infected branches or stems contain tiny black pimplelike spore bodies which produce orange to dark red spore masses called spore tendrils or spore horns. Wood beneath the canker is often stained light brown. Little mortality is caused by this disease. Cankers also may be found in association with wounds or natural openings in the bark. This canker is not considered a vigorous parasite on healthy trees, but can hasten the death of trees injured or weakened by other agents, such as weather, insects, or other diseases.

Figure 11 — Orange-colored *Cytospora* canker on a small aspen.

Figure 12 -- Spore tendrils of *Cytospora chrysosperma.*

E. Cryptosphaeria canker, caused by *Cryptosphaeria populina* (Pers.) Sacc., is widespread throughout the West. Cankers are associated with bark wounds on live branches or stems. After infection occurs, the fungus moves into the sapwood and then into the heartwood. Cankers are usually long and narrow, from 2 to 4 inches wide and up to 10 feet or more in length. Bark around cankers is light brown to orange and the tree forms an annual callus in response to the infection. The dead, black, stringy, sootylike bark adheres tightly to the sapwood and contains small, light-colored areas in contrast to sooty-bark canker. Branch cankers are often found on large trees where they girdle the branch and enlarge onto the trunk. The sexual form, *Libertella* spp., is associated with extensive stain, decay, and mortality in aspen and other poplars; wounded trees of all ages can be attacked.

Figure 13 — *Cryptosphaeria* canker on aspen.

Figure 14 — Bark removed from *Cryptosphaeria* canker to show sooty-bark and light-colored bark specks.

A. ***Ciborinia whetzelii*** (Seaver) Seaver, causing "ink spot" on aspen leaves, occurs intermittently throughout the aspen type. Damage by this disease varies from none to extensive defoliation. At several year intervals, the disease can reach localized epidemic proportions in aspen stands. Epidemics of this disease coincide with warm, moist, spring and summer weather. During dry years, little or no evidence of this disease is seen. Symptoms of the disease are easily recognized by examination of foliage from an infected tree. Leaves have circular black spots of sclerotia appearing on them in early summer. The spots fall out either while the leaves are still on the tree or after the leaves have fallen. Frequently, premature defoliation of all or a major portion of the tree occurs. Severe, repeated defoliations by ink spot and black leaf spot *(Marssonina populi)* may cause mortality of smaller trees, although there is considerable variation in the susceptibility of different clones to these diseases.

Figure 15 — Ink spot on aspen leaves.

B. ***Marssonina populi*** (Lib.) Magn. causes black leaf spot on aspen. This foliage disease is similar to ink spot in its erratic occurrence from year to year. Generally, in late summer, the fungus produces small, circular tan leaf spots which later turn black. Borders of the lesions are yellow or gold. Often, premature defoliation will occur on all or a major portion of the tree. Infected leaves often are reduced in size and affected trees may appear bronzed from a distance. Leaf spot on aspen causes little mortality and the merchantability of infected trees is not affected by this disease. However, severe defoliation for 2 years has caused mortality and branch dieback of susceptible clones.

Figure 16 — Aspen leaves infected with black leaf spot *(Marssonina).*

C. ***Pollaccia radiosa*** (Lib.) Bald. & Cif., *(Venturia tremulae),* causes a leaf and twig blight commonly called "shepherd's crook." Black spots first appear on the leaves in early or mid-May. These spots gradually enlarge until the leaf is killed; the fungus then spreads down the petiole causing dieback, curling, and the blackening of new terminal shoots. Seedlings and sprouts are primarily affected, resulting in the death and deformation of their terminal growth. Secondary infections appear late in May on shoots and leaves until most shoots and leaves of terminal growth are infected. Repeated infections can reduce increment growth and cause stem distortion.

Figure 17 — Shepherd's crook of aspen.

D. ***Melampsora medusae*** Thuem. Is the most common aspen leaf rust in the West. A conifer-aspen rust, this fungus needs an alternate host such as Douglas-fir, pine, true firs, spruce, or hemlock to complete its life cycle. In the spring, infected aspen leaves on the ground release spores that infect the alternate host. Wind disseminated spores from the alternate host then infect aspen leaves in the summer, producing yellow spots and orange-yellow spores which reinfect more leaves. Premature defoliation may occur on heavily infected trees, but damage to the trees is not considered serious.

Figure 18 — *Melampsora* leaf rust of aspen.

References

Anderson, Gerald W., Thomas E. Hinds, and Donald M. Knutson. 1977. Decay and discoloration of aspen. USDA Forest Service, Forest Insect and Disease Leaflet 149, 4 pp., Washington, D.C.

Anderson, Gerald W., and Ralph L. Anderson. 1968. Relationship between density and quaking aspen and incidence of Hypoxylon canker. Forest Science 14:107-112.

Anderson, Neil A., Michael E. Ostry, and Gerald W. Anderson. 1979. Insect wounds as infection sites for *Hypoxylon mammatum* on trembling aspen. Phytopathology 69:476-479.

Anderson, Ralph L. 1964. Hypoxylon canker impact on aspen. Phytopathology 54:253-257.

Anderson, R. L., and G. W. Anderson, revised by Arthur L. Schipper, Jr. 1979. Hypoxylon canker of aspen. USDA Forest Service, Forest Insect and Disease Leaflet 6, 7 pp., Washington, D.C.

Anderson, Robert L., and Arthur L. Schipper, Jr. 1978. A system for predicting the amount of *Phellinus (Fomes) igniarius* rot in trembling aspen stands. USDA Forest Service Research Note NC-232, 4 pp., North Central Forest and Range Experiment Station, St. Paul, Minn.

Andrews, Stuart R., and Wallace E. Eslyn. 1960. Sooty-bark canker of aspen in New Mexico. Plant Disease Reporter 44:373.

Boyce, John Shaw. 1961. Forest pathology. McGraw-Hill Book Company, New York. 572 pp.

Brinkman, Kenneth A., and Eugene I. Roe. 1975. Quaking aspen: silvics and management in the Lake States. Agriculture Handbook 486, 52 pp., U.S. Department of Agriculture, Washington, D.C.

Davidson, Ross W., and Thomas E. Hinds. 1956. Hypoxylon canker of aspen in Colorado. Plant Disease Reporter 40:157-158.

Davidson, Ross W., Thomas E. Hinds, and Frank G. Hawksworth. 1959. Decay of aspen in Colorado. U.S. Department of Agriculture, Forest Service. Rocky Mountain Forest and Range Experiment Station, Station Paper 45, 14 pp. Fort Collins, Colo.

Hepting, G. H. 1971. Diseases of forest and shade trees of the United States. U.S. Department of Agriculture Handbook No. 386. 658 pp.

Hinds, Thomas E. 1962. Inoculations with the sooty-bark canker fungus on aspen. Plant Disease Reporter 46:57-58.

Hinds, Thomas E. 1963. Extent of decay associated with *Fomes igniarius* sporophores in Colorado aspen. USDA Forest Service Research Note RM-4, 4 pp., Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

Hinds, T. E. 1964. Distribution of aspen cankers in Colorado. Plant Disease Reporter 48:610-614.

Hinds, T. E. 1972a. Ceratocystis canker of aspen. Phytopathology 62:213-220.

Hinds, T. E. 1972b. Insect transmission of *Ceratocystis* species associated with aspen cankers. Phytopathology 62:221-225.

Hinds, T. E. 1976. Aspen mortality in Rocky Mountain campgrounds. USDA Forest Service Research Paper RM-164, 20 pp. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

Hinds, T. E., and R. G. Krebill. 1975. Wounds and canker diseases on western aspen. USDA Forest Service, Forest Pest Leaflet 152, 9 pp. Washington, D.C.

Hinds, Thomas E., and Eugene M. Wengert. 1977. Growth and decay losses in Colorado aspen. USDA Forest Service Research Paper RM-193, 10 pp. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

Hinds, Thomas E. 1981. Cryptosphaeria canker and Libertella decay of aspen. Phytopathology 71:1137-1145.

Juzwik, J., W. T. Nishijima, and T. E. Hinds. 1978. Survey of aspen cankers in Colorado. Plant Disease Reporter 62:906-910.

Krebill, R. G. 1972. Mortality of aspen on the Gros Ventre elk winter range. USDA Forest Service Research Paper INT-129, 16 pp. Intermountain Forest and Range Experiment Station, Ogden, Utah.

Landis, T. D., and A. K. Evans. 1974. A relationship between *Fomes applanatus* and aspen windthrow. Plant Disease Reporter 58:110-113.

Leaphart, C. D. 1963. Armillaria root rot. USDA Forest Service, Forest Pest Leaflet 78, 8 pp. Washington, D.C.

Lindsey, J. Page, and Robert L. Gilbertson. 1978. Basidiomycetes that decay aspen in North America. 406 pp. J. Cramer, Lehre, West Germany.

Livingston, C. H., T. E. Hinds, C. E. Seliskar, and R. E. Klein. 1979. A "drooping" malady of western aspen. Plant Disease Reporter 63:923-927.

Mielke, James L. 1957. Aspen leaf blight in the Intermountain Region. USDA Forest Service Research Note 42, 5 pp. Intermountain Forest and Range Experiment Station, Ogden, Utah.

Ohman, J. H., and K. J. Kessler, Jr. 1964. White trunk rot of hardwoods. USDA Forest Service, Forest Pest Leaflet 88, 7 pp. Washington, D.C.

Riffle, Jerry W., and Thomas E. Hinds. 1969. Hypoxylon canker of aspen in New Mexico and Arizona. Plant Disease Reporter 53:454.

Ross, William D. 1976a. Fungi associated with root diseases of aspen in Wyoming. Canadian Journal of Botany 54:734-744.

Schipper, Arthur L. Jr., and Robert L. Anderson. 1976. How to identify Hypoxylon canker of aspen, 5 pp. U.S. Department of Agriculture, North Central Forest Experiment Station, St. Paul, Minn.

Walters, James W., Thomas E. Hinds, David W. Johnson, and Jerome Beatty. 1982. Effects of partial cutting on diseases, mortality, and regeneration of Rocky Mountain aspen stands. USDA Forest Service Research Paper RM-240, 12 pp. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

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